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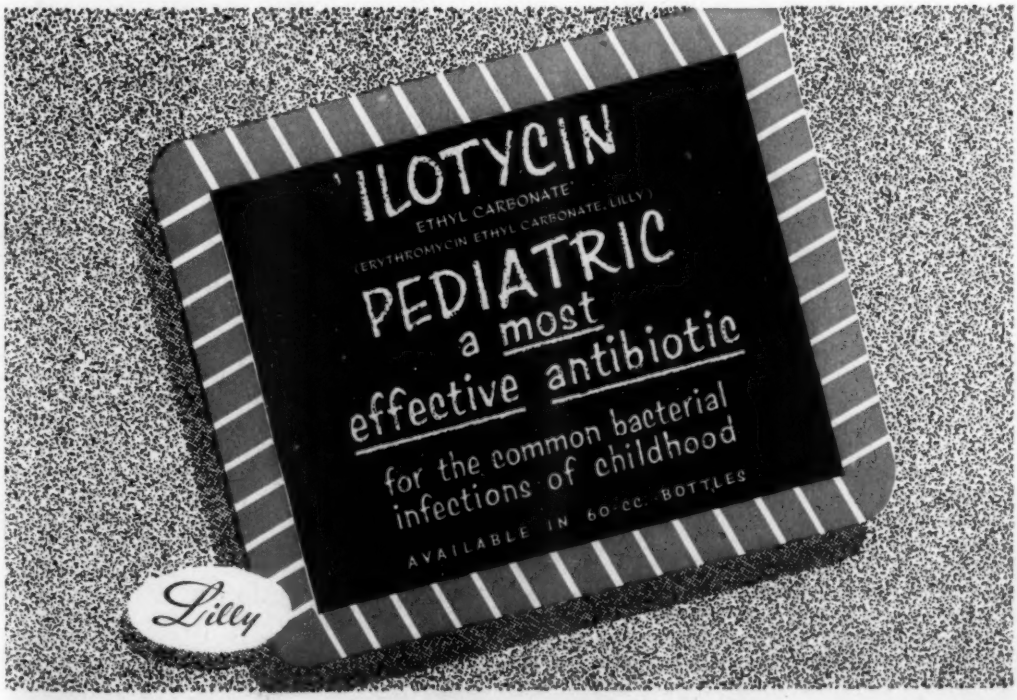

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De Rebus Medicis Et Politicis

BY ROBERT B. HOMAN, JR., M. D., EL PASO, TEXAS
MEMBER OF THE HOUSE OF DELEGATES OF THE AMERICAN MEDICAL ASSOCIATION

PHYSICIAN - HOSPITAL RELATIONSHIP

The two most important "organizations" in the medical profession are the County Medical Society and the medical staff of the hospital. Of these, of course, the most important is the county society, for membership in the society is the primary pre-requisite for appointment to the staff of any or all hospitals. In view of this fact, one is inclined to wonder why the majority of doctors, having attained membership, completely ignore the basic organization of the profession. To put it briefly, attendance at county society meetings is disgracefully poor.

More or less compulsory attendance is required, on the other hand, at hospital staff meetings. Despite this compulsion, or perhaps because of it, there has been some misunderstanding regarding hospital - physician relationship in many areas. In order to let you know what is going on, the following points of agreement between the A. M. A. and the American Hospital Association are filched from *The President's Page*, (J. A. M. A., Vol. 153, No. 4, P. 433). The principles of this joint report are:

GENERAL PURPOSE

"1. The general purpose of hospitals and physicians is to aid each other in the delivery of the best possible medical care to patients. To attain such a purpose requires full cooperation among medical staffs, governing boards and administrative heads of hospitals. One important method of attaining this objective is that duly designated representatives of the medical staff shall have free and direct access to the governing board with due consideration to the position of the administrator as chief executive officer of the hospital. The various methods by which the medical staff may have access to the hospital governing board follow.

"a. The executive committee of the medical staff and a committee of the govering board with the hospital administrator can serve as a joint committee.

"b. Representatives of the medical staff can serve as members of the medical staff committee of the governing board with the hospital administrator.

"c. Representatives elected by the medical staff can attend meetings of the hospital governing board.

"d. Members of the medical staff can be members of the hospital governing board.

"2. The professional evaluation of chiefs of service and members of the medical staff should be the responsibility of the medical profession. The method of selection of these individuals must be subject to local arrangements and local conditions. In any such arrangement, however, the principle of the freedom of the staff to make recommendations, subject to the approval of the hospital governing board should be recognized.

"3. The medical profession and the hospitals recognize that certain special services, such as anesthesiology, pathology, radiology, and physical medicine, are integral parts of the practice of medicine and of the services necessary for hospital patients. Physicians in these fields should have the professional status of other members of the medical staff. Chiefs in these specialties must assume also the administrative responsibilities and relationships customarily associated with such positions.

INDIVIDUAL TERMS

"4. The right of an individual to develop the terms of his services on the basis of local conditions and needs is recognized but such contractual arrangements should in all cases ensure (a) the policy of professional incentive for the physician, and (b) progressive development of the hospital departments involved, in order that increasingly improved services to patients may be rendered. Moreover, a physician shall not dispose of his professional attainments or services to any hospital, lay body, organization, group, or individual, by whatever name called, or however organized, under terms or conditions which permit exploitation of the patient, the hospital, or the physician.

"5. The chief of a hospital department may have access to financial information regarding his department.

"6. It is desirable that means should be provided at local, state and national levels

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APHORISMS AND MEMORABILIA TRUTHS AND CONCEPTS CONCERNING THE CARDIO-VASCULAR SYSTEM

By Andrew M. Babey, M. D., Las Cruces, N. M.

(Continued)

21. "With complete cardiac arrest, which has gone on for 30 seconds in the presence of convulsions, it is obvious that intramuscular and subcutaneous injections will do nothing, and one should administer adrenalin by intracardiac route. For that reason, we like to have, at the bedside of patients who are in a very actue stage of the Adams-Stokes syndrome, a sterile syringe filled with Adrenalin with a long needle."—Dr. Conger Williams, *Am. Pract. Sept.* 1952, *p.* 757.

22. "If we could only select with certainty those few cases which do respond we should be very pleased to continue recommending sympathectomy, but unfortunately all the preoperative tests to improve selection which have been tried, seem to have failed utterly. We must reluctantly admit that a large number of quite useless operations have been performed and that there is very little prospect of any satisfactory improvement in surgical technique or selection in the near future."—John McMichael, *British Med. Journal, May,* 3, *953 *p.* 936.

23. "Looked at more analytically, the data show clearly that established essential hypertension is a serious disease in young people and especially in young men, not so serious in middle aged people of both sexes, and a mere annoyance in the elderly. More specifically according to Bechaard, a man who develops definite essential hypertension between the ages of thirty and forty-nine is eight times more likely to die within ten years of onset than is a comparable normotensive man over the same period of time. Women, in contrast, who develop the disease between thirty and forty-nine, are only 1.4 times more likely to die during such a period of time than are comparable normotensive women. Men and women who develop the disease between the ages of fifty and sixty-nine have an excellent chance of living out a normal life span and attempts to treat the disease in this age group, except in unusual cases, are often ill-advised. These data suggest that overtreatment may have been the rule rather than the exception in the management of many cases of essential hypertension but they underscore the urgent need for effective therapy of the disorder, particularly when it occurs in young people and especially in young men." — Carelton Chapman, *Minn. Med. Oct.* 1952, *p.* 951.

24. "In three cases of this series, the clinical picture was unmistakably that of bacterial endocarditis, except that cultures of the blood failed to reveal any organisms. Combined therapy, consisting of 5,000,000 to 10,000,000 units of penicillin per day administered by intermittent intramuscular or intravenous route, together with dehydrostreptomycin, was given from four to six weeks in these cases. The infection was controlled in all three. Treatment in cases of this type should be the same as used in cases in which the organism is either penicillin resistant or is an enterococcus. Apparently these patients in whom sterile cultures of blood persist do not do as well when smaller daily doses of penicillin are given."—Joseph Geraci (Mayo Clinic) *Minn. Med. Sept* 1952, *p.* 865.

25. "If the patient and physician wish to employ a rational form of prophylaxis capable in animals and man of slowing or even reversing deposition of cholestorol in the arterial intima, they will be limited to a diet low in cholesterol, fat and calories, adequate in protein, and in suitable cases supplemented with thyroid." — William Dock, *Am. Journal of Med. March* 1952. *p.* 358.

26. "I think that at the present time one can say that, while adding lipotropic agents to the diet is reasonable and is not likely to hurt the patient, the use of these factors as a substitute for a proper diet would be a mistake." — William Dock, *loc. cit.*

27. "And this brings us to a fundamental question: At the ages at which myocardial infarction is likely to occur, is it worth while going on a diet just to postpone this sort of accident? Many people drive their cars dangerously, quite well aware of the risk. Dangerous occupations and dangerous sports are followed. Why not dangerous dining? I think it is unnecessary for everybody to go through life on a diet free of ice-cream and omelet, just to prolong his existence." — William Dock, *loc. cit.*

28. "Makes one wonder whether it is worth while, at the time when your children are able to take care of themselves, for you to devote yourself to a way of living which you do not like, merely to try to prolong that tiresome exsistence for a greater span of years. Each person will have to make up his mind whether his obligations and inclinations make it worth while for him to follow such a regimen. Perhaps most people will make the some decision as some of my medical friends. They are convinced that the theory is correct, but they see no reason why they should adhere to the practice." — William Dock, *loc. cit.*

29. "With nocturnal dyspnea, patients jump out of bed and sit up on the edge of the bed, or go to the window to get some air. They think the fresh air helps them, but stale air would have done just as well. It was getting out of bed with their feet down that made the difference. Patients with nocturnal dyspnea ought not to be put to bed to begin with. I am as certain as I am alive that I have slain a number of patients years ago by putting them to bed. For years I have been in the habit of keeping cardiacs out of bed, from the start, even those in congestive failure. Then came the next step, using chair treatment for myocardial infarction; first sporadically and for the past several years as a routine, unless they are too feeble to sit up or are in shock. I see no advantage to having a coronary case stay in bed. The only advantage is to the doctor's reputation, not to the patient's welfare, because if a cardiac dies in bed we are all forgiven. If he should die in a chair the doctor will get the rap, until enough time has elapsed so that the patients will accept this procedure as a standard method of therapy. We have sold the bill of goods that they have to be in bed and they resent any advice to get out. Frequently, when I have told the family that I would like to get this man out of bed and have him sit in a chair, they would refuse to do it. Maybe they won't in future years." — Samuel Levine, *Am. Practitioner, Dec.* 1952 *p.* 1013.

30. "Considering the time and effort that have been given to electrocardiology, the harvest of information has not been excessive. Its principal practical use is in detecting myocardial ischaemia — an achievement which, while satisfying the physician's very proper preoccupation with exact diagnosis, sometimes gives the patient a load of anxiety without conferring any noteworthy therapeutic benefit. The time is coming, however, when electrocardiography will find its proper level as a faithful servant and cease to be an exacting master." — The Lancet, *Sept.* 20, 1952, *p.* 569.

31. "I propose to continue to operate upon a proportion of hypertensive patients. I select them myself. I refuse those who are symptomless, those who have sever cardiac disease, and those who have cerebral palsies. The oldest in my series was 54, which is probably too old; and in a doubtful case I would give the chance of operation to a man rather than a woman, though some men are very depressed after the second side is done. Women do better on any treatment. I am not always right, but then who is? What matters is that I naturally select patients for operation with all the earnestness and sense of responsibility of which I am capable."— Sir James Learmonth, *The Lancet, Nov.* 11, 1950, *p.* 508.

32. "Patent Ductus Arteriosus:

. . . The fact that the continuous murmur seldom develops before 1 year of age and often not until the patient is 3 or 4 years of age, means that the diagnosis prior to this time is difficult. Fortunately in most instances the condition causes little or no disability. Therefore in the vast majority of patients, early diagnosis is not necessary. Occasionally, however, a large patent ductus arteriosus may cause great difficulty in early infancy. Therefore an infant with great cardiac enlargement and evidence of excessive pulmonary blood flow who has a balanced electro-cardiogram and has only a systolic murmur and a mid-diastolic murmur but no continuous murmur, should be sent to a specialist for study." — Helen B. Taussig, *Circulation, Vol.* 6, *Dec.* 1952, *p.* 930

33. "The most characteristic of all signs is notching of the ribs. Notching of the ribs may be seen as early as three years of age, but it is usually not apparent until 12 or 14 years of age. Its occurrence in adults is the rule. Indeed absence of notching of the ribs in an adult who has clinical evidence of coarctation of the aorta should always suggest the possibility of coarctation of the abdominal aorta. Under such circumstances either angiocardiography or aortography is indicated to determine the location of the constriction."—H. Taussig, *loc. cit. p.* 931.

34. "In tetralogy of Fallot, the more intense the cyanosis, the greater is the patients incapacity, and the fainter is the murmur. A very cyanotic patient may have no murmur, but if there is a minimal cyanosis and no murmur it is not a tetralogy of Fallot." — H. Taussig, *loc. cit. p.* 934.

35. "Pure Pulmonary Stenosis.

. . . The presence or absence of cyanosis depends upon whether or not the foramen ovale is completely sealed. If the foramen

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DR. SCHUESSLER ELECTED NEW SOUTHWESTERN MEDICAL ASSOCIATION PRESIDENT

Dr. Willard W. Schuessler of El Paso was elected president of the Southwestern Medical Association at the organization's annual meeting in Tucson, October 29 through 31. Dr. Wesley O. Connor, Jr., of Albuquerque was the retiring president.

Born in Tinley Park, Chicago, Dr. Schuessler was graduated from Terrill Preparatory School in Dallas in 1927 and from Southern Methodist University in 1931. He received his M. D. from Baylor Medical College in 1934. He was a member of Phi Chi Medical Fraternity; Alpha Pi Alpha honorary medical fraternity; and Alpha Omega Alpha, honorary medical fraternity. He was awarded the Surgeon General's Award in 1934.

Dr. Willard W. Schuessler

Dr. Schuessler interned at Baylor University Hospital in Dallas and was resident surgeon at the Baylor University Hospital. Between 1936 and 1946 he was attending plastic surgeon at Baylor University Hospital, Parkland Hospital in Dallas, and the Texas Crippled Children's Hospital in Dallas.

He was associate professor of plastic surgery at Baylor University Hospital and Southwestern Medical School until 1946.

During his service with the Army he was chief of plastic surgery at Brooks General Hospital in San Antonio from 1940 to 1942; chief of plastic surgery at Walter Reed Hospital in Washington, D. C. from 1942 to 1944; instructor in plastic surgery at the Army Medical College in Washington, D. C. from 1942 to 1944; and chief of plastic surgery at William Beaumont Hospital from 1944 to 1946.

Dr. Schuessler was awarded the Legion of Merit in 1946. William Beaumont General Hospital presented him with a Certificate of Achievement.

At present he is attending plastic surgeon at Southwestern General Hospital, Providence Memorial Hospital, Hotel Dieu and El Paso General Hospital in El Paso. He is plastic surgeon consultant at William Beaumont Army Hospital and La Tuna Federal Correctional Institute.

He is a member of the American Association of Plastic Surgeons, the American Society of Plastic and Reconstructive Surgery, the International College of Surgeons, the Texas Surgical Society and the founder group of the Southwestern Surgical Congress.

CLINICAL SESSION PLANNED FOR GENERAL PRACTITIONERS

Have you made your plans to attend the seventh annual Clinical Session of the American Medical Association in St. Louis, Dec. 1-4? More than 3,500 physicians will be there.

A program has been designed to give the general practitioner an opportunity to see and hear the latest developments in medicine. More than 150 papers by outstanding physicians will cover such topics as internal medicine, surgery, pediatrics, obstetrics and gynecology, tuberculosis and other diseases of the chest, cardiovascular diseases, arthritis, dermatology, gastrointestinal diseases and neuropsychiatry.

GOUT *

By Richard D. Haines, M. D., Temple, Texas

Although gout has afflicted mankind for centuries, it is often misdiagnosed. Unfortunately, it is infrequently considered as a diagnostic possibility. There has been little concrete data available until recently to clarify the underlying abnormal physiology prevailing in gouty patients. Now, with a better understanding of uric acid metabolism and the effects of available uricosuric agents, two major steps have been made in the problem of gout. Our purpose is to collect some of these facts and to correlate them into the present accepted plan of therapy.

CLINICAL PICTURE

The incidence of gout is difficult to determine; however, its relative frequency is sufficient to warrant its consideration in each instance of joint disease. The clinical picture is usually diagnostic, and close questioning of the patient for salient features of the joint process will make the diagnosis in a large group of patients previously diagnosed as having "chronic arthritis" or "rheumatism." Classically, gout can be divided into two, at times, distinct forms — acute recurring gout, and chronic gouty arthritis. The onset of the acute attack is sudden, and often begins during the early morning hours. The pain is usually severe, and will reach its maximum intensity within a few hours. Attacks are, therefore, precipitously incapacitating, and the joints exhibit local swelling, warmth, erythema, and marked limitation of motion. Desquammation often follows the subsidence of the local joint signs. In mild cases, the attacks may last for from one to seven days, and the disease process may be limited to only one joint. As the attacks recur, the local joint manifestations persist longer, and frequently multiple joints are involved. Some patients have infrequent attacks years apart, while others have frequent recurring attacks and progressive joint disability—progressing to the stage of gouty arthritis. Other patients actually never reach the clinical stage of gouty arthritis. When the stage of gouty arthritis occurs, the joint distress may not subside, pain and tenderness persist, swelling continues, and joint deformity and disability ensue.

The diagnosis of gout can be confused with osteoarthritis; atypical rheumatoid arthritis; palindromic rheumatism; occasionally, rheumatic fever; and intermittent hydroarthrosis. During the gouty arthritic stage, the disease is often mistaken for osteoarthritis or rheumatoid arthritis. Here it is the "life history" of gout that is most important. Characteristics of the early attacks should be inquired about, especially regarding the asymptomatic interval periods. With the exception of palindromic rheumatism, freedom of joint disability between acute attacks is almost pathognomonic of gout. In this diagnostic decision, the duration of attacks is paramount for attacks of palindromic rheumatism usually last only a few hours, while an untreated acute attack of gout usually lasts for days. The history of acute attacks of gout precipitated by trauma, surgery, diuretics, hypervitaminosis, hyperalimentation, or alcoholism are factors to be noted. The presence of tophi; and, when available, the finding of hyperuricemia are added diagnostic features. Lastly, the therapeutic effectiveness of colchicine is most important.

DIFFERENTIAL DIAGNOSIS OF GOUT

	Gout	Osteoarthritis	Rheumatoid Arthritis	Palindromic Rheumatism
Sex	Males	Equal	Females	Equal
Onset	Dramatic	Insidious	Usually slow	Sudden
Pain	Severe	Mild	Variable	Usually marked
Course Disease	Recurring acute attacks	Slowly progressive	Progressive disability	Little progression, recurring attacks
Duration Attack	Days	Not attacks	Not attacks	Hours
Interval History	Freedom from distress	Progressive, slowly	Progressive	Freedom from distress
Laboratory Studies	Elevated uric acid, sed-rate variable	None diagnostic	Sed-rate elevated	None diagnostic
Therapy Response	Excellent to Colchicine	Not specific	Colchicine no effect, Cortisone variable	Not specific
Roentgenographic Findings	Late "punched out" areas	Bony spurs, lipping	Osteoporosis, soft tissue swelling	None

PATHOPHYSIOLOGY

The specific, underlying metabolic abnormality in gout has not been clearly defined. Recent evidence has indicated some alteration in the normal pituitary adrenal inter-relationships, and this has placed gout among the ever increasing list of "diseases of adaptation." [1] Other important investigation has been conducted pertaining to uric

* From the Department of Internal Medicine of the Scott and White Clinic.

acid metabolism in normal and gouty patients with resulting insight into the genesis of uric acid as well as to its response to various therapeutic agents usually dispensed in the treatment of gout.

Attention was directed toward the adrenal gland by Talbott, et al,[2, 3] when evidence was presented to the effect that gouty patients have electrolyte imbalance similar to that found in adrenal insufficiency. Later, Wolfson and co-authors[4, 5] proposed that this alteration is a diminution in ACTH release rather than a primary adrenal deficiency. This observation is based on the absence of increased adrenal cortical activity in acute gout.

Other investigators have studied gouty patients with emphasis on hematological, hormonal, electrolytic, and uric acid determinations and have not found any concrete, consistent evidence of increased adrenal cortical function in acute gout.[6] From a practical clinical standpoint, acute gout is beneficially altered by ACTH; however, attacks may occur shortly after its discontinuance. The specific pathophysiological change to account for this "dual" effect is still speculative.

CHANGING CONCEPT

Employing isotope techniques, there is a changing concept regarding the relationship of acute gout and uric acid. With little reservation, the manifestations of chronic gouty arthritis are due to the urate deposits in the tissues; however, in acute gout, this relationship is unconvincing. Clinically, one is sometimes confronted with acute attacks of gout associated with a normal serum uric acid level. Also, other disease states associated with hyperuricemia fail to develop clinically acute gout. The proposal has been made that acute gout is precipitated by some, as yet, unidentified precursor of uric acid rather than uric acid, per se.[7]

Recent isotope studies have revealed that various substances, such as dietary ammonia, glycine, serine, and carbon dioxide are potential precursors of uric acid.[8, 9, 10, 11, 12, 13] Also, uric acid can be formed from purines of nucleic acid.[14, 15] Generally speaking, "the amount of uric acid formed in the body depends upon the rate of the purines formed from their precursors, the availability of the precursors, the amount of direct oxidation of the formed purines, the turn over rates of the pentose nucleic acids of different cells, and the rate of regeneration of the cells."[16]

This theoretical data is a practical application as one must consider dietary carbohydrates, proteins, and fats as potential precursors of uric acid, and this information evaluated when dietary restrictions are instituted.

CONTROVERSY

The controversy still continues regarding the explanation of elevated blood uric acid. Theoretically, three possibilities exist; namely, increased production of uric acid, decreased destruction of uric acid, and altered excretion. Many of the gouty patients are in the age group in which one expects to find renal damage present; and, therefore, conclusive information pertaining to altered excretion is difficult to obtain. Data is available showing that all the urate in the plasma is in a filterable form; and in normal individuals, five to six milligrams of urate are filtered each minute. Due to the tubular urate reabsorption mechanism, probably less than 10 per cent is excreted in the urine. Friedman and Byers[18] demonstrated increased urate excretion in young gouty patients as compared with normal individuals. Yu[19] also cites evidence of increased urate excretion in gouty individuals; however, in another series, when compared with renal function tests, the urate excretion was reduced in individuals with renal damage. Although it is not convulsive, Berliner[20] suggests that in gout there is "more likely an increase in urate production than a primary renal abnormality, but this is by no means certain." It appears that on a low purine diet excretion of uric acid is greater in gouty patients than in normal individuals. This is particularly true before renal damage occurs as renal damage aggravates uric acid retention.

Studies directed toward measuring body uric acid values have been carried out. The "miscible pool" of uric acid is that quantity of uric acid which is capable of promptly mixing with intravenously administered isotopic uric acid. The value for a non-gouty male is approximately 1200 milligrams.[21] Gouty patients have been studied and they have a greatly expanded miscible uric acid pool.[21] Urate in the body can be viewed as existing in three compartments; that in the plasma, that in the body water, and that in a solid form (tophi). Experimental data[21] suggests that urate recently deposited in tophi is capable of mixing with injected isotopic uric acid and is also capable of resolution. This last statement has practical clinical significance as it reflects the possibility of uricosuric agents for decreasing the urate deposits in chronic gouty patients.

THERAPY

If there is debate regarding the genesis of gout, there is reasonable agreement regarding its treatment. In brief, therapeutically speaking, gout has three phases; acute gout, interval gout, and chronic gouty arthritis. One is often prone to emphasize

the treatment of the acute attacks and be little concerned regarding the interval treatment, which in many instances is relatively more important, preventing, if possible, the undesired crippling effects of chronic tophaceous gouty arthritis.

The principles of treatment are quite flexible for interval gout, but rather rigid and dogmatic in acute gout.

OUTLINE OF THERAPY FOR GOUT

ACUTE ATTACKS
1. Low purine diet
2. Colchicine
3. Enteric coated aspirin

ACUTE ATTACK FAILING TO RESPOND
ACTH or Cortisone added

INTERVAL GOUT — INFREQUENT ATTACKS
1. Low purine diet
2. Treat each attack as acute attack

INTERVAL GOUT — FREQUENT ATTACKS
1. Low purine diet
2. Benemid
3. Enteric coated aspirin — if intolerant to Benemid

TOPHACEOUS GOUTY ARTHRITIS
1. Purine free diet
2. Benemid
3. Colchicine

Because of the severity of the attack, a diagnosis must be established promptly when possible. However, if diagnosis is indeterminate in an acute monarticular attack, the effectiveness of colchicine may be used as a diagnostic measure. The patient should be hospitalized. If no allergy is known, enteric coated aspirin (75-80 gr. in divided doses within the first 24 hours) and simultaneous colchicine (1/200 gr. tablets every two hours until the onset of diarrhea) are promptly instituted. These two medications, plus the use of a purine free diet will serve to alleviate 90 per cent of acute attacks of gout. Those cases failing to subside within 24 hours are treated with the addition of ACTH or Cortisone and the temporary discontinuance of colchicine. We have relied on Cortisone (150-200 mg. per 24 hours until the attack subsides) and on the day before Cortisone withdrawal, reinstitution of colchicine (1 table, four times a day for 4 to 6 days) — hoping that the patient is below the diarrhea threshold, and also hoping to prevent an acute flare-up following Cortisone withdrawal. At least, one of the mechanisms by which aspirin, ACTH, and Cortisone produce beneficial results is by producing uric acid diuresis. On the other hand, colchicine is ineffective in uric acid diuresis; yet, it is the drug of choice in treatment of an acute attack of gout.

With the alleviation of the acute attack, the patient is placed in the category of "interval gout" and is so treated whether this be interval gout with recurring acute attacks or gouty tophaceous arthritis, per se. The rigidity of the program depends on the patient's willingness to follow instructions, the frequency and severity of attacks, and the extent of the joint disability.

Realizing that future investigation may drastically alter the dietary restrictions, for practical purposes, the patients are generally instructed in a low purine diet. The exception is for the individual with frequent severe attacks or incapacitating tophaceous gouty arthritis, and then rigid dietary restrictions are made. For the individual with infrequent mild attacks, prompt treatment of each attack and only moderate dietary restrictions seem adequate. The patient with frequent attacks is also instructed in the use of Benemid (2 gr. daily in divided doses). This has proved to be a satisfactory maintenance plan of therapy for an increasing number of patients. Aspirin and Benemid are not simultaneously prescribed as there is evidence that they are physiologically antagonistic. Those individuals intolerant to Benemid are maintained on enteric coated aspirin, 80 grains daily, for the first six days, and the fifteenth to twenty-first of each month.

Patients with gouty arthritis of severe degree are treated in a like manner; yet, a few cases seem resistent to this program and, therefore, colchicine, ordinarily reserved for the treatment of acute attacks, is also administered in subdiarrheic doses once a week.

In summary, the clinical picture of gout has been reviewed, its important diagnostic features stressed, altered uric acid and hormonal imbalances mentioned, and a practical plan of therapy suggested.

REFERENCES

1. Selye, Hans: *Textbook of Endocrinology*, Montreal, Canada, Acta Endocrinologica, Universite de Montreal, 1947.
2. Talbott, J. H.; Jacobson, B. M.; and Oberg, S. A.: Electrolyte Balance in Acute Gout, J. Clin. Investigation 14:411-421, (July) 1935.
3. Talbott, J. H., and Coombs, F. S.: Metabolic Studies on Patients with Gout, J. A. M. A. 110:1977-1982, (June 11) 1938.
4. Wolfson, W. Q., and Cohn, C.: The Role of the Pituitary Adrenal Corticotrophic Hormone (ACTH) and of Adrenal Cortical Steroid Hormones in Pathological Physiology and Experimental Therapeutics of Clinical Gout, in *Proceedings of the First Clinical ACTH Conference, edited* by J. R. Mote Philadelphia, The Blakiston Co., 1950, p. 241.
5. Wolfson, W. Q., and others: ACTH and Colchicine in Clinical Treatment of Acute Gouty Arthritis: Physiological Considerations and Review of Therapeutic Results in 51 Attacks, J. Michigan M. Soc. 49:1058-1064; 1083, (Sept.) 1950.
6. Levin, M. H., and others: Metabolic Studies in Gout, J. Clin. Endocrinol. 12:506-518, (May) 1952.
7. Gutman, A. B. in Combined Staff Clinic: Uric Acid Metabolism and Gout, edited by F. K. Heath, Am. J. Med. 9:799-817, (Dec.) 1950.
8. Barnes, F. W., Jr., and Schoenheimer, R.: On Biological Synthesis of Purines and Pyrimidines, J. Biol. Chem. 151:123-139, (Nov.) 1943.
9. Sonne, J. C.; Buchanan, J. M.; and Delluva, A. M.: Biological Precursors of Uric Acid; Role of Lactate, Acetate, and Formate in Synthesis of Ureide Groups of Uric Acid, J. Biol. Chem. 173:69-79, (March) 1948.
10. Buchanan, J. M.; Sonne, J. C.; and Delluva, A. M.: Biological Precursors of Uric Acid; Role of Lactate, Glycine,

and Carbon Dioxide as Precursors of Carbon Chain and Nitrogen Atom 7 of Uric Acid, J. Biol. Chem. 173:81-98, (March) 1948.
11 Shemin, D., and Rittenberg, D.: On Utilization of Glycine for Uric Acid Synthesis in Man, J. Biol. Chem. 167-875-876, (March) 1947.
12 Karlsson, J. L., and Barker, H. A.: Biosynthesis of Uric Acid Labeled with Radioactive Carbon, J. Biol. Chem. 177:597-599, (Feb.) 1949.
13. Elwyn, D., and Sprinson, D. B.: Role of Serine and Acetate in Uric Acid Formation, J. Biol. Chem. 184:465-474, (June) 1950.
14. Hammarsten, E., and Hevesy, G.: Rate of Renewal of Ribo - and Desoxyribo Nucleic Acids, Acta Physiol. Scandinav. 11:335-343, 1946.
15. Furst, S. S.; Roll, P. M.; and Brown, G. B.; On Renewal of Purines of Desoxypentose and Pentose Nucleic Acids, J. Biol. Chem. 183:251-266, (March) 1950.
16. Shemin, David in Combined Staff Clinic: Uric Acid Metabolism and Gout, edited by F. K. Heath, Am. J. Med. 9:799-817, (Dec.) 1950.
17 Berliner, R. W.; Hilton, J. G.; Yu, T. F.; and Kennedy, T. J., Jr.: Renal Mechanism and Urate Excretion in Man, J. Clin. Investigation 29:396-401, (April) 1950
18. Friedman, M., and Byers, S. O.: Increased Renal Excretion of Urate in Young Patients with Gout, Am. J. Med. 9:31-34, (July) 1950.
19. Yu, T. F in Combined Staff Clinic: Uric Acid Metabolism and Gout, edited by F. K. Heath, Am. J Med. 9:799-817, (Dec.) 1950.
20. Berliner, R. W. in Combined Staff Clinic: Uric Acid Metabolism and Gout, edited by F. K. Heath, Am. J. Med. 9:799-817, (Dec.) 1950.
21. Stetten, DeWitt, Jr. in Combined Staff Clinic: Uric Acid Metabolism and Gout, edited by F. K. Heath, Am. J. Med. 9:799-817, (Dec.) 1950.

Aphorisms

(Continued From Page 406)

ovale is completely sealed, there is no cyanosis. If, however, there is probe patency of the foramen ovale, the high pressure in the right auricle will force the valve covering the foramen ovale to open and a right-to-left shunt will be established. Although cyanosis of this origin may be present at birth it usually develops insidiously and becomes manifest between 2 and 6 years of age."—H. Taussig, *loc. cit. p.* 935.

36. "German measles in the first trimester of pregnancy is associated with a high incidence of congenital malformations of the heart, congenital cataracts and microcephaly in the offspring. The cardiac abnormality is the least serious of these three complications. The resultant malformation is frequently a patent ductus arteriosus. Microcephaly is a terrible tragedy. The earlier the infection occurs, the greater is the risk of injury to the fetus. If the infection occurs during the first month of pregnancy, the chance of a defective offspring is nearly 100 per cent. After the third month, German measles seldom if ever injures the fetus."—H. Taussig, *loc. cit. p.* 939

37. "In most instances a malformation with a loud murmur carries a better prognosis than one in which there is no murmur. Indeed the absence of a murmur is generally indicative of an extremely serious malformation. Under such circumstances if the lung fields are excessively clear, early operation is usually indicated." — H. Taussig, *loc. cit. p.* 940.

Cancer Specialist to Speak in El Paso

Dr. Alexander Brunschwig, one of the world's foremost cancer surgeons, will speak before El Paso County Medical Society, El Paso Tumor Clinic and lay workers of the El Paso Chapter of the American Cancer Society when he arrives in El Paso Nov. 24.

Dr. Brunschwig, who was born in El Paso, has instituted new surgical methods for cancers heretofore considered inoperable. He has authored several books on cancer surgery, including "Radical Surgery in Advanced Cancer of the Abdomen," and "Surgery of Pancreatic Tumors."

Speaking engagements for Dr. Brunschwig in El Paso will begin with a luncheon for lay workers of the local chapter of the American Cancer Society, followed by an address to members of the El Paso Tumor Clinic at El Paso General Hospital at 1 p. m. Nov. 24. He will speak to the El Paso County Medical Society that evening.

Dr. Brunschwig was born in El Paso on Sept. 11, 1901, the son of Mr. and Mrs. Felix Brunschwig. He was graduated from the University of Chicago and Rush Medical College and did post-graduate work in Strasbourg and Paris.

Following service in the U. S. Navy he began his medical practice in Chicago in 1928 as a member of the University of Chicago Clinics, and was named professor of surgery at that school in 1940. He is now a member of the surgical staff and head of the gynecology department at Memorial Hospital for Treatment of Cancer and Allied Diseases in New York and professor of clinical surgery at the Cornell University College of Medicine.

Dr. Brunschwig has been named an honorary member of the surgical societes of Austria, Guatemala, Czechoslovakia, and France. He has been decorated with Medal of Charles U. of Prague, and the White Lion of Czechoslovakia, which followed his assignment to Unitarian Service Committee of Teaching Missions to that country in 1946.

He now maintains offices at 444 East 68th Street in New York and his home is in Pelham, N. Y.

BENTYL HYDROCHLORIDE OR DICYCLOMINE EFFECTIVE IN RELIEVING ANGINA PECTORIS

By Robert J. Antos, M. D., Phoenix

Bentyl Hydrochloride* is a parasympatholytic agent having its greatest action as a smooth muscle antispasmodic affecting the gastroenteric and genito-urinary tracts. It apparently exerts the beneficial effects of the belladonna drugs without the undesirable effects.[1, 2, 3] Up to now, most of the investigational work with this drug has been directed toward the bowels and ureters. The recent reports of other autonomic blocking agents, particularly their effects on the circulatory system, have stimulated my interest in this drug along this line.

A few years ago there appeared several papers on the sympathetic blocking agent tetraethylammonium.[4, 5] This drug was shown to have a vagal blocking influence,[6] and a fleeting ability to reduce blood pressure by producing a transient peripheral vasodilatation.[7] The vasodilatation produced by this drug is effective in normal people as well as in compensated and decompensated cardiacs.[8] A few reports appeared where this drug was dangerous to give in the presence of generalizd arteriosclerosis with heart disease,[9] although the drug has been advocated as a diagnostic tool prior to sympathectomy in hypertension.[10]

ATROPINE

The atropine group of drugs in small doses stimulate, and in large doses, depress smooth and cardiac muscles.[11] Outside of this group of drugs there has appeared no work on the action of parasympatholytic agents on the cardiovascular system.[12] When Bentyl became available it quickly gained use in relieving smooth muscle spasm. It was noted that in many patients who were known to have angina pectoris, Bentyl not only relieved the intestinal spasm, but it diminished greatly the angina. This led me to wonder if there were some action in this drug to relieve anginospasm. Thus the following study was born.

METHOD OF STUDY: The instrument used in this study is the Krasno Ivy Flicker Photometer. This is a device which has the facility of emitting a flickering light, the frequency of which is controlled by the operator.

The principle of the device is discussed in the paper by Krasno and Ivy.[13] An excellent discussion of flicker fusion principles is given by Landis.[14] Suffice it to say that the flicker fusion point or threshold (F. F. T.) varies with the rate of flow through retinal vessels. Thus if the vessel is dilated and it carries a greater volume of blood (and thereby oxygen) the ability of the retina to see a flickering light is increased. In short, the retina detects a flicker at a higher frequency when the volume of blood (blood vessel caliber, is increased.

ORIGINAL METHOD

In the original method of Ivy the frequency is determined before and after a standard dose of glyceryl trinitrate. An increase of more than sixty cycles in the flicker fusion threshold indicated presence of angiospasm.

In this study the same method was applied using Bentyl in place of the glyceryl trinitrate. The results are given in the tables. The procedure briefly was as follows:

The frequency at which the flicker could be detected was determined. Bentyl was given by mouth. Then the flicker frequency was determined again at thirty minute intervals. The results are in Table I. On those patients displaying angiospasm on their original test using glyceryl trinitrate, a second test was made after they had been given Bentyl by mouth for several days.

THREE GROUPS

The subjects were divided into three groups as follows:

1. A group of normal, healthy adult males and females who displayed no evidence of coronary disease. These were mostly student nurses and orderlies ranging in age from seventeen to thirty-nine years.
2. Persons with angina pectoris. These were later divided into two groups as follows:
   a. Those displaying evidence of angiospasm with the Flicker Photometer.
   b. Those displaying no angiospasm by the Flicker Photometer.

* "Bentyl Hydrochloride" is the trade-mark of the Wm. S. Merrell Company for its brand of dicyclomine.

The results of the experiment are tabulated as follows:

EFFECT OF BENTYL ON HEALTHY YOUNG ADULTS WITHOUT EVIDENCE OF CORONARY DISEASE (according to method of Roback, Krasno and Ivy)[15]

TABLE I

Pt.	Age & Sex	Bentyl Dosage	Flicker Fusion Change 15 min.	30 min.	1 hr.	2 hr.	Effect on F. F. T.
1	17 Fe.	100 mg.	+10	−15	+5	+10	No Effect
2	20 Fe.	100 mg.	0	0	+5	0	No Effect
3	20 Male	100 mg.	+5	+5	+30	+10	No Effect
4	36 Fe.	100 mg.	0	0	+10	+10	No Effect
5	21 Male	100 mg.	0	+40	+65	+40	Increased*
6	18 Fe.	100 mg.	0	0	0	0	No Effect
7	19 Fe.	100 mg.	0	0	+5	0	No Effect
8	21 Fe.	100 mg.	+5	+10	+5	−5	No Effect
9	19 Fe.	100 mg.	0	0	0	+5	No Effect
10	#5 retested at rest		0	0	+5	−5	No Effect

* This male orderly was tested after working the night shift. He was not rested and apparently had angiospasm from fatigue.

From the foregoing tabulation of the results of testing nine supposedly normal, healthy adults it is concluded that Bentyl had no effect on the flicker fusion threshold in the dosage given. In one case (#5) the subject was tested after a night of work, and the spasm present in this subject was affected by Bentyl. When this same subject was retested at rest, the response was negative.

The next step was to test the effects of Bentyl on patients with angina pectoris. The results in some cases were encouraging and in others disappointing. After juggling the results about, significant facts could be deduced when these subjects were divided into two groups; those that displayed angiospasm by way of the flicker fusion test, and those who did not display angiospasm.

The tabulation at bottom of the previous column is from trials made from a series of patients with angina who displayed evidence of angiospasm.

The next table gives the results on the same group of patients after they had been taking Bentyl for periods ranging from one week to several months:

Pt.	Bentyl Dosage	Time on Bentyl	F.F.T. after Bentyl	Change in F.F.T.	Glyceryl trinitrate needed to control pain	Clinical change
1	50 mg. t.i.d.	5 wks.	+40	−50	None	Much improved but complained of dizziness
2	50 mg. t.i.d.	4 wks.	+30	−45	1 to 2 weekly	Very much improved — dizziness
3	50 mg. t.i.d.	6 wks.	+60	−100	None	Very much improved
4	10 mg. t.i.d.	8 wks.	+20	−60	None	Very much improved
5	100 mg. t.i.d.	2 wks.	?	?	None	Felt much better
6	25 mg. t.i.d.	1 mo.	+40	−25	None	Improved, dizziness, varicose veins hurt
7	25 mg. t.i.d.	6 wks.	+60	−65	None	Improved, dizziness, varicose veins hurt
8	10 mg. t.i.d.	8 wks.	+50	−20	one daily	Mod. improved — slight nausea
9	10 mg. t.i.d.	8 wks.	+30	−55	None	Much improved throbbing in arms
10	20 mg. t.i.d.	7 wks.	+45	−20	None	Improved — slight dizziness
11	50 mg. t.i.d.	3½ wks.	+25	−75	Occasional	Much improved — blurred near vision
12	25 mg. t.i.d.	2 wks.	+30	−35	None	Improved — dryness in mouth

From the foregoing results it is concluded that all of the patients who showed evidence of angiospasm (F. F. T. change above 60) were benefitted by the effects of Bentyl administered in doses ranging from 10 mg. t. i. d. to 100 mg. t. i. d.

Following are the results on a few patients who had angina and did not show evidence of angiospasm:

Pt.	Age & Sex	Glyceryl trinitrate used	F.F.T. change	Bentyl dosage	Glyceryl trinitrate after Bentyl	F. F. T. change	Reactions & subjective changes
1	46 Fe.	None	+10	100 mg. t. i. d.	None	+10	very slight improvement - dizzy
2	71 Male	None	+40	100 mg. q. i. d.	None	+45	No improvement – nausea
3	57 Male	1 to 2 daily	−10	100 mg. q. i. d.	1 to 2 daily	+5	Felt dopey while taking pills
4	67 Fe.	3 to 4 daily	+15	100 mg. q. i. d.	3 to 4 daily	+5	No improvement – nausea
5	80 Male	1 to 2 daily	+5	100 mg. q. i. d.	3 to 4 daily	0	No improvement – nausea
6	64 Male	None	+35	100 mg. q. i. d.	None	+10	No improvement – blurred near vision
7	68 Fe.	1 to 2 daily	+20	100 mg. q. i. d.	1 to 2 daily	0	No improvement – blurred near vision
8	72 Fe.	None	−5	100 mg. q. i. d.	None	+5	No improvement – nausea

CONTROL READINGS OF ANGINAL PATIENTS WITH ANGIOSPASM AS INDICATED BY FLICKER PHOTOMETER

Pt.	Age & Sex	F. F. T. Increase	Degree of angina	Glyceryl trinitrate used to relieve pain
1	68 Fe.	+90	Mod. severe on slight exertion	Occasionally — 3 to 4 times weekly
2	63 Fe.	+75	Mod. severe on slight exertion	1 to 3 daily
3	68 Fe.	+160	Very severe even at rest	10 to 12 daily
4	35 Fe.	+80	Mod. on exertion	3 to 5 daily
5	71 Fe.	+200	Severe even at rest	None - used alcohol in tsp. doses 6 to 8 times daily. Nitroglycerine gave severe headache.
6	35 Fe.	+65	Mild on exertion	2 to 3 daily
7	40 Fe.	+125	Mild on exertion	None – just stopped and rested
8	71 Male	+70	Moderate	3 to 4 daily
9	49 Male	+85	Moderate	1 to 2 daily
10	57 Male	+65	Mild to moderate	None – rested
11	51 Male	+90	Moderate - daily	2 to 3 daily
12	65 Fe.	+65	Mild	2 to 3 weekly

It should be mentioned that the patients in the preceding group took little or no glyceryl trinitrate because they got no benefit from it.

CONCLUSIONS

1. Bentyl did not have any measurable effect on the flicker fusion threshold of normal, healthy adults who displayed no angina.
2. Bentyl had no effect on angina in those people with angina who displayed no angiospasm as demonstrated by the Krasno-Ivy Flicker Photometer.
3. Bentyl did have a beneficial effect on angina in patients who did display angiospasm.
4. Bentyl is a useful drug in the treatment of reduced coronary flow when it is caused by spasm of the artery, but Bentyl is of no value in overcoming the reduced flow when the coronary vessels are narrowed by arteriosclerosis. In those patients benefitted by Bentyl, the amount of nitroglycerine needed to control pain was greatly reduced, and in some cases no glyceryl trinitrate was needed at all.
5. Bentyl has an action on the coronary vessels similar to that of glyceryl trinitrate although not so rapid in onset. The effect lasts longer and in many people with mild angina it can replace glyceryl trinitrate altogether.
6. In many of the cases the Bentyl was synergistic with glyceryl trinitrate. The results with the flicker photometer indicate that these changes are not mental suggestion: a vaso-dilatation is produced.
7. It can be surmised from the results that patients with angina who get little or no relief from glyceryl trinitrate will get little benefit from Bentyl. On the other hand, those patients who do get relief from glyceryl trinitrate should get good results from Bentyl. If the Bentyl alone will give relief, the patient can avoid the headache or throbbing that comes from trinitrate. If they must use trinitrate, they will use much less if used in conjunction with Bentyl.
8. The maximum dose was 100 mg. q. i. d. At this dose almost all patients observed undesirable side reactions. The average dose seems to be in the neighborhood of 25 to 50 mg. t. i. d.
9. Caution is indicated in elderly people with arteriosclerosis. The higher than usual antispasmodic doses (10 mg.) will produce atropine-like effects. Those patients with marked varicose veins might develop recurrence of long-forgotten leg pains.

REFERENCES

1. Hufford, Jour. Mich. Med. Soc., *49*: 1308, 1950.
2. Hock, J. A. M. A., *40*:22, 1951.
3. Chamberlain, Gastroenterology, 17:224, 1951.
4. Pearl, Ann. Surg., *128*:1092, 1948.
5. Ibid., *128*:1100, 1948.
6. Biorck and Ejrup, Acta. Med. Scandinav., *133*:382, 1949.
7. Steinberg, Anesthesiology, *10*:428, 1949.
8. Epstein, J. A. M. A., *139*:685, 1949.
9. Lindgren and Frisk, Acta. Med. Scandinav., *131*:581, 1948.
10. Frisk et al, Amer. J. Med., *5*:807, 1948.
11. Sollman, A Manual of Pharmacology, W. B. Sanders Co., 1950.
12. Ausman, D. C. and Arneth, J. J., Wisconsin Med. Jour., Nov. 1951.
13. Krasno and Ivy, Circulation, *1*:1267, 1950.
14. Landis, Scientific Monthly, *LXXIII*:308, 1951.
15. Roback, Krasno and Ivy, J. Appl. Physiol., *4*:566, 1952.

From Medical Grand Rounds*

These notes are abstracts of opinions expressed by staff members during case presentations at the Medical Grand Rounds of the Pratt Diagnostic Clinic, Boston.

Wilson's disease has long been considered a degenerative disease (hepatolenticular degeneration). Although not congenital, it is familial in the sense that more than one member of a generation is usually affected. A disease of young people, it is characterized by the development and progression of a movement abnormality that has been described as a wing-beating tremor. Rigidity, and occasionally choreoathetosis, may be prominent. Because of the rigidity, difficulty in speaking and swallowing is common, and emotional overreaction (spastic smiling) usually occurs. There is an associated hepatic cirrhosis, which may be evident early in the course of the disease or may only be demonstrable by abnormal values in liver function tests. The diagnosis may be clinched by finding the pathognomonic Kayser-Fleischer ring, which is a greenish brown-to-golden ring of pigmentation near the limbus of the cornea. Recent work suggests that this condition may be related to an abnormality of copper metabolism: it has been found that there is an excessive excretion of copper in the urine of these patients; and when BAL (British anti-lewisite) is given, there is a further significant increase in the excretion of copper, followed by improvement in the patient's condition.

Physician – Hospital Relationship

(Continued From Page 405)

for review of problems of individual hospital-physician relationship by organized medical and hospital groups."

These principles are applicable only after the local county society and the individual hospital staff have approved them. The medical profession will write the ticket if the doctors assert their views through the county society and the hospital staff.

Perhaps your attendance and interest are more important than you think.

BONE COCCIDIOIDOMYCOSIS
CASE REPORT - CURED BY SURGERY

By Herbert E. Hipps, M. D., Waco, Texas

A review of the literature on disseminated coccidioidomycosis indicates that it is a severe disease with a mortality rate of around 50 per cent and that bone lesions seldom if ever get well.

Tremble[3] states, "A cure for the disseminated or granular form of the disease has not been discovered." He further states ..."with dissemination the prognosis is grave." Fraser[2] states, "Amputation has often been advised and has proved to be of benefit in single lesions. Rest is the treatment of choice for the present, until some chemo-therapeutic anti-biotic agent....has been perfected." The literature as a whole abounds with pessimistic case reports and statements like these on the curability of bone coccidioidomycosis.

However Foley[1] reports a simple lesion of the skull, treated by wide radical excision that did get well.

The following case is reported because the patient's lesion in the tibia healed completely following radical surgical treatment and has remained well for three years.

CASE REPORT — R. C. AGE 7, WHITE

This child was seen first in my office November 6, 1948, with a swollen painful left knee with several draining sinuses over the front of it. The knee first became involved eleven months before. He was treated in a hospital with rest, penicillin and streptomycin for three months with no benefit. In April 1947 a biopsy was done and a diagnosis of coccidioidomycosis was made. The biopsy wound broke down and began to drain. Additional sinuses have since formed around the biopsy scar and all are draining. He has always been in good health. His parents state that he has never had any serious respiratory disease. This child lived in the San Joaquin Valley of California for a two year period of time prior to the onset of his knee disability.

EXAMINATION

This child's general condition was good. His lungs were normal. His left knee was in an aluminum splint. It was swollen. He could flex it only about fifteen degrees. Pain occurred with motion. There were several draining sinus openings over the front of the knee. A thick yellowish-white purulent material came from them. His oral temperature was 99.6 degrees.

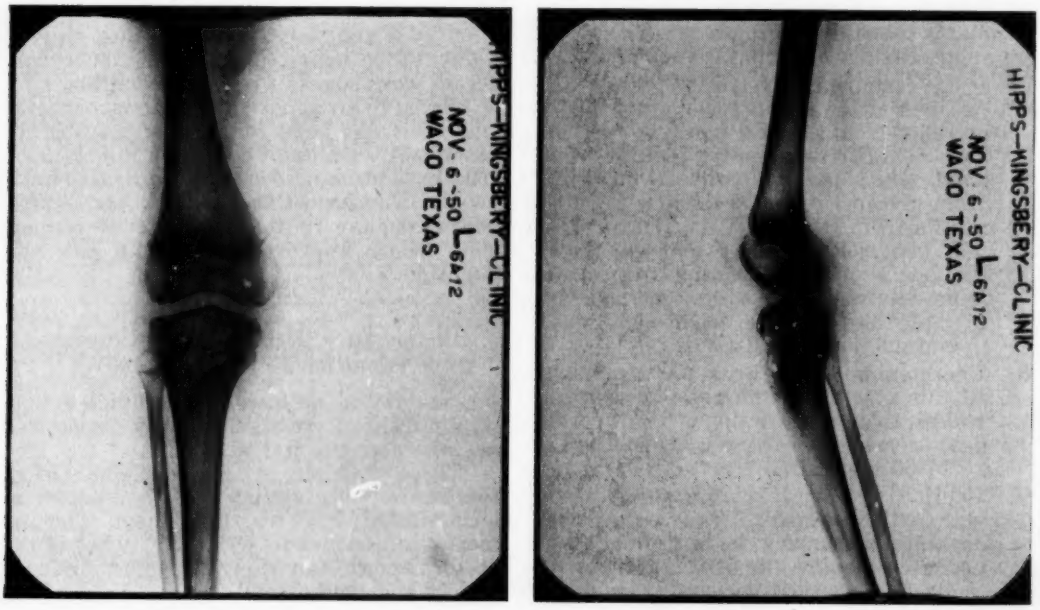

FIGURE 1.—An a.p. and lateral x-ray of the tibia when first seen.

X-RAY REPORT

There is a multilocular cystic-like destructive process involving the upper end of the tibia. It was present both above and below the epiphyseal line, but did not involve the joint surface. There was no new bone formation.

A chest x-ray made at the hospital was reported as being negative.

LABORATORY REPORTS

Urine analysis — negative
Hb — 78 per cent.
Rbc — 4,500,000
Wbc — 6,300
Lymph — 56 per cent
Polys — 44 per cent

Smear and culture from sinus drainage — mixed growth of staphylococcus, streptococcus, and Gram-negative rods resembling B. Coli.

Cultures on Sabouraud Dextrose Agar after two weeks indicate—Coccidioidomycosis imites —

Cultures on Lowenstein Media — no acid fast bacilli grown in six weeks.

Coccidioidin skin tests — In dilution of 1-10,000 questionably positive.

In dilution of 1-1000 positive, exhibiting an area of induration over one cm. in diameter.

TREATMENT

This patient was in the hospital two separate periods of time. He was treated conservatively the first time, surgically the second. Because sensitivity tests indicated that his mixed infection was resistent to penicillin and streptomycin, he was given orally large doses of Terramycin. In addition, through a catheter extending down into the cystic cavity, Aureomycin and Terramycin in an aqueous Zephiran solution was injected every eight hours. The knee was continually immobilized in a metal splint. This treatment program was carried out for six weeks.

He was sent home with a bulky dressing on the knee. He remained home one month and returned to my office for the dressing to be changed. On removal of the dressing one small sinus was found to have closed but all the other were draining as much as ever. A smear showed the Gram-negative rod to be present, but no staphylococci or streptococci.

For the next thirty days his knee was dressed at intervals of three or four days to allow the surficial skin irritation to clear up. He was then re-admitted to the hospital and the operation as follows was done.

OPERATION

A vertical, slightly curved incision following some of the sinus openings was made. The skin, soft tissue, and periosteum were retracted and some bone was removed, widely exposing the cavity in the tibia. Part of the tibial tubercle was lifted up to gain wide access to the cavity. The knee joint was not opened. This cavity, including the soft tissue about it, was cleansed thoroughly with a detergent soap and water, dried, then cleansed with ether. Then a 10 per cent iodine solution was puddled in the cavity covering every part of it, including the raw edges of the bone that had been cut away. This solution remained in contact with the bone for ten minutes. When removed the entire bone surface was stained thoroughly a dark brown color.

Now with an osteotome, gouge, and curette the entire bone surface inside the cavity was cut away for a distance of about one or two millimeters all around. The cavity, including the soft tissue was then filled with a solution of 1-1000 aqueous Zephiran. The tibial tubercle was replaced, sutured down with chromic O and the wound was closed. Considerable difficulty was encountered in closing the skin, but by undermining it, and doing a Z plasty it was closed together tightly. All sinus tracts were freshened and closed. No drain tube was used. A bulky dressing was applied and the leg was replaced in the metal splint.

FIGURE 2.—An a.p. and lateral x-ray of the tibia when last seen.

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POST-OPERATIVE CARE

The stitches were removed in two weeks. Slight drainage had occurred. On the next dressing in two more weeks there was considerable drainage; but this continued to decrease with each succeeding dressing and in two months time the wound dried over and has remained dry ever since. His leg remained immobilized for another month after all draining ceased and he continued to remain in bed.

After this period of time he was allowed up with crutches, bearing part weight on the leg. In another two months full weight bearing was permitted. The last time he was seen was February 19, 1952. He still had no drainage, walked without a limp, had a full range of motion in the knee, had gained weight and seemed to be well.

A recent letter from his mother states that he has had no more trouble with it.

SUMMARY AND CONCLUSION

A case report of coccidioidomycosis imites of the tibia is reported that did get well following radical surgical-chemotherapeutic treatment.

In view of this case and the case report of Foley, it seems likely that some of these patients can be cured by a radical surgical attack.

BIBLIOGRAPHY

(1) Foley, M. P et al — Coccidioidal Granuloma West. J. Surg. Obst. & Gyn. Dec 1940.
(2) Fraser, Chas. G. — Coccidioidomycosis and Tuberculosis in same Bones. Ann. Surg., 133: 116-122, Jan. 1951.
(3) Tremble, H. G. — Coccidioidomycosis Dis. Chest., 20: 586-610 Dec. 1951.

New Mexico GP's Meet in Roswell

The regular annual meeting of the New Mexico Chapter of the American Academy of General Practice, postponed since May, will be held in Roswell, New Mexico, Saturday, November 14.

The meeting had first been planned for the annual meeting of the New Mexico Medical Society in Albuquerque in May but because of an extra House of Delegates meeting, the General Practice session was postponed.

In Roswell the annual business session will precede a program to be presented by speakers from Southwestern Medical School in Dallas. Luncheon and coffee breaks will be served. The registration fee will be $10. All physicians are invited to attend. Official credit for post-graduate training will be granted for the total number of hours of attendance.

Texas Internists to Meet

Next meeting of the Texas Academy of Internal Medicine has been set for Saturday and Sunday, Dec. 5 and 6. The meeting will be held at the Shamrock Hotel in Houston where each of you will be expected to make your own hotel room reservations. We are looking forward to a very fine program and at some subsequent date a copy of the program will be mailed to each of you.

At the general request of the membership a post-graduate course in endocrinology, particularly on the pituitary-adrenal aspects, is also scheduled to be held in Houston on Wednesday and Thursday, Dec. 2 and 3. As guest speaker for this program, which will be held in cooperation with the Baylor University College of Medicine, will be Dr. Jerry Conn, professor of medicine at the University of Michigan. This course will be open only to members of the Texas Academy of Internal Medicine at a nominal fee of $10.00 to those attending. We feel certain that this will be an extremely worthwhile course.

On the interval date, Friday, Dec. 4, the Texas Rheumatism Association meeting will also be held at the Shamrock Hotel, with Dr. Ed Boland of Los Angeles as the guest speaker. By payment of a registration fee of $5.00 any member of the Texas Academy of Internal Medicine will also be allowed to attend this program.

DRUG REACTIONS

The Influence Of Drugs On Blood And Bone Marrow

Heck, F. J., Postgrad. Med. 13:185, 1953

Drug-induced granulocytopenia, thrombocytopenic purpura, hemolytic anemia, aplastic or hypoplastic anemia and leukemia-like disorders are discussed. One important point to remember is that blood dyscrasia due to drugs may develop long after treatment is initiated or during a second course of therapy. Bone marrow changes may not immediately be reflected in peripheral blood so that red and white counts can not be relied upon to give the earliest possible warning. Constant vigilance is required to prevent disastrous results. Drugs known to depress the hematopoietic system should be used only if no other agent is available and then only after considering the "calculated risk."

Mayo Clinic

Clinical Clippings, May. 1953.